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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/562,778	07/27/2006	Zhonglin Chai	2354/370	7943

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NIXON PEABODY LLP - PATENT GROUP
1100 CLINTON SQUARE
ROCHESTER, NY 14604

EXAMINER	
HADDAD, MAHER M	
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1644	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/562,778	CHAI ET AL.	
	Examiner	Art Unit	
	Maher M. Haddad	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *18 August 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-32* is/are pending in the application.

4a) Of the above claim(s) *1-9,17-20 and 22-32* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *10-16 and 21* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☒ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *12/29/05*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. Claims 1-32 are pending.

2. Applicant's election with traverse of Group VII, claims 10-16 and 21 drawn to a method for treating or preventing a condition related to synthesis of an ECM protein, the method including modulating the expression and/or activity of a CDA, wherein the condition is fibrosis and the species of kidney fibrosis, filed on 08/18/08, is acknowledged.

Applicant's traversal is on the grounds that all groups are closely related and can be searched without undue burden. This is not found persuasive because Applicant's inventions do not contribute a special technical feature when viewed over the prior art they do not have a single general inventive concept and so lack unity of invention as set forth in the previous Office Action.

The requirement is still deemed proper and is therefore made FINAL.

3. Claims 1-9, 17-20 and 22-32 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to nonelected inventions.

4. Claims 10-16 and 21 are under examination as they read on to a method for treating or preventing a condition related to synthesis of an ECM protein, the method including modulating the expression and/or activity of a CDA, wherein the condition is fibrosis and the species of kidney fibrosis.

5. Applicant's IDS, filed 12/29/05, is acknowledged, however, Ozbun et al, reference no. 1 was crossed out because said reference is duplicate of Ozbun et al reference cited on PTO-892, mailed 4/17/08.

6. claim 10 is objected to because it fails to indicate what does CDA stand for.

7. The following is a quotation of the second paragraph of 35 U.S.C. 112.

> *The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.*

8. Claims 10-16 and 21 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

A. The claims are incomplete for omitting essential steps and ingredients. While all of the technical details of a method need not be recited, the claims should include enough information to clearly and accurately describe the invention and how it is to be practiced.

The minimum requirements for method steps minimally include a contacting step in which the reaction of the sample with the reagents necessary for the assay is recited.

9. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

10. Claims 10-16 and 21 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The specification does not reasonably provide enablement for a method for treating or preventing a condition related to synthesis of an ECM protein, the method including modulating the expression and/or activity of a CDA in claim 10, wherein the condition is fibrosis in claim 11, wherein the fibrosis is due to a burn, a heart attack, treatment with a chemotherapeutic drug, exposure to radiation, or surgery in claim 12, wherein the fibrosis is major organ fibrosis fibrosis in claim 13, wherein the major organ is kidney, wherein the major organ fibrosis is due to a condition selected from the group consisting of diabetes, hypertension, viral hepatitis, alcohol abuse, macular degeneration, retinal retinopathy and vitreal retinopathy in claim 15, wherein the condition is renal fibrosis as a result of diabetes in claim 16, wherein the CDA is CDA1 in claim 21. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention.

The specification on page 7, lines 1-7 discloses that there has been no previous report on CDA1 expression in the kidney, applicants have shown that CDA1 is expressed in the kidney including distal tubules and collecting ducts. CDA1 expression in the distal tubules and collecting ducts of normal rats demonstrated both cytoplasmic and nuclear patterns. CDA1 was rarely expressed in the glomeruli in the normal rat kidney. The specification on page 7 lines 12-20 discloses that co-localisation of CDA1 expression with fibrosis in the remnant kidney following subtotal nephrectomy (STNx) was also observed (Fig. 3). CD1 expression appeared in the podocytes in the glomeruli following renal mass reduction (Fig. 6A) which was not seen I the normal kidney. Cytoplasmic and nuclear staining patterns were evident, particularly in the sclerotic glomeruli

and at sites of tubulointerstitial fibrosis. Our preliminary data also indicate that therapeutic approaches which block the action of angiotensin II such as an AT1 receptor antagonist, valsartan, are associated with less cell injury and with attenuation of CD1 expression (Fig. 3B). The specification on page 7, lines 26-31 discloses that an increase in the production of matrix proteins including collagen IV and fibronectin in the CDA1 transfected cells when compared with cells transfected with vehicle alone. This finding indicates that CDA1 may be directly linked to the production of extracellular matrix, and subsequently to the development of renal fibrosis.

"The amount of guidance or direction needed to enable the invention is inversely related to the amount of knowledge in the state of the art as well as the predictability in the art." In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970). The "amount of guidance or direction" refers to that information in the application, as originally filed, that teaches exactly how to make or use the invention. The more that is known in the prior art about the nature of the invention, how to make, and how to use the invention, and the more predictable the art is, the less information needs to be explicitly stated in the specification. In contrast, if little is known in the prior art about the nature of the invention and the art is unpredictable, the specification would need more detail as to how to make and use the invention in order to be enabling (MPEP 2164.03)." The MPEP also states that physiological activity can be considered inherently unpredictable. Further, in Rasmusson v. SmithKline Beecham Corp., 75 USPQ2d 1297-1303 (CAFC 2005), the court states "If mere plausibility were the test for enablement under section 112, applicants could obtain patent rights to "inventions" consisting of little more than respectable guesses as to the likelihood of their success. When one of the guesses later proved true, the 'inventor' would be rewarded the spoils instead of the party who demonstrated that the method actually worked. That scenario is not consistent with the statutory requirement that the inventor enable an invention rather than merely proposing an unproved hypothesis."

While "modulating the expression and/or activity of a CDA" may have a notion of "inhibiting" the function of the claimed CDA; there is insufficient biochemical or structural information to enable the skilled artisan to make and use the antagonist that "modulating the expression and/or activity of a CDA", as broadly claimed. "It is not sufficient to define the recombinant molecule by its principal biological activity, e.g. having protein A activity, because an alleged conception having no more specificity than that is simply a wish to know the identity of any material with that biological property." Colbert v. Lofdahl, 21 USPQ2d, 1068, 1071 (BPAI 1992).

Further, besides SEQ ID NO: 2, the specification fails to disclose any CDA or CDA1. CDA is an arbitrary name. While the name itself may have some notion of the activity of the protein, there is nothing in the claims which distinctly claims the protein. For example, others in the field may isolate the same protein and give it an entirely different name such as DENTT (see Ozbun et al of record). Claiming biochemical molecules by a particular name given to the protein by various workers in the field fails to distinctly claim what that protein is and of what compositions comprising that protein are made. Further, it is not clear whether the CDA is a human, cat, rat, mouse, among other species.

The functional activities of claimed "modulating the expression and/or activity of a CDA", are mutually exclusive in that they reach opposing endpoints, and in that they employ structurally distinct *agonists* or *antagonists* to accomplish these mutually exclusive endpoints. The skilled artisan would not have a reasonable expectation that the same compound that produce inhibition of CDA would also serve to produce activation of CDA. One skilled in the art at the time of the invention would not be able to predict which compound will "stimulate/promote/inhibit" the expression and/or activity of a CDA. Consequently the skilled artisan would not know how to use the instant invention as broadly claimed. Applicant is relying upon certain biological activities to support an entire genus of compounds that modulate the expression and/or activity of a CDA. The claims as written encompass a broad genus of compounds with an unlimited number of possibilities.

On the basis of the disclosed co-localisation of CDA1 expression with fibrosis in the remnant kidney following subtotal nephrectomy observation alone (see page 7, lines 12-13), applicant concludes that the scope of the a compound that modulate the expression and/or activity of a CDA encompassed by the claimed invention can have biological activity to treat or prevent a condition related to synthesis of an ECM protein including kidney fibrosis and be provided as pharmaceutical compositions to subjects including human to effectively treat/prevent fibrosis including kidney fibrosis. The specification contemplated that Angiotensin II antagonist such as an AT-1 receptor antagonist, e.g., valsartan, or TGFβ antagonist or CTGF antagonist can be used to treat/prevent a condition related to synthesis of an ECM protein (fibrosis disorder). No such agents were produced or tested, it is unclear if these assay results are predictive of treating or preventing a condition related to synthesis of an ECM protein.

The lack of any working examples is exacerbated because the invention is in a highly unpredictable art-treatment/prevention of fibrosis- and while the level of skill of in the art may be high, the state of the prior art is that it is in fact unknown and untested what are the underlying physiologic bases of the therapeutic effects of a method for modulating the expression and/or activity of a CDA. Although CDA1 expression was increased by TGF β1 *in vitro* (see pg 8, lines 30-31), its actual function and mechanisms of activation are unclear. Moreover, it is not known whether modulating the expression and/or activity of a CDA interventions can treat/prevent fibrosis.

While the specification is not enabled for both treatment and prevention, the burden of enabling the prevention of a disease (i.e. the need for additional testing) would be greater than that of enabling a treatment due to the need to screen those mammals susceptible to such diseases and the difficulty of proof that the administration of the drug was the agent that acted to prevent the condition. Further, the specification does not provide guidance as to how one skilled in the art would go about screening those patients susceptible to kidney fibrosis within the scope of the presently claimed invention. Nor is sufficient guidance provided as to a specific protocol to be utilized in order to prove the efficacy of the presently claimed modulating the expression and/or activity of a CDA in preventing fibrosis.

Reasonable correlation must exist between the scope of the claims and scope of the enablement set forth. In view on the quantity of experimentation necessary the limited working examples, the nature of the invention, the state of the prior art, the unpredictability of the art and the breadth of the claims, it would take undue trials and errors to practice the claimed invention.

11. Claims 10-16 and 21 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Applicant is not in possession of a method for treating or preventing a condition related to synthesis of an ECM protein, the method including modulating the expression and/or activity of a CDA in claim 10, wherein the condition is fibrosis in claim 11, wherein the fibrosis is due to a burn, a heart attack, treatment with a chemotherapeutic drug, exposure to radiation, or surgery in claim 12, wherein the fibrosis is major organ fibrosis fibrosis in claim 13, wherein the major organ is kidney, wherein the major organ fibrosis is due to a condition selected from the group consisting of diabetes, hypertension, viral hepatitis, alcohol abuse, macular degeneration, retinal retinopathy and vitreal retinopathy in claim 15, wherein the condition is renal fibrosis as a result of diabetes in claim 16, wherein the CDA is CDA1in claim 21.

Neither the exemplary embodiments nor the specification's general method appears to describe structural features, in structural terms, that are common to the genus. That is, the specification provides neither a representative number of species that modulate the expression and/or activity of a CDA to describe the claimed genus, nor does it provide a description of structural features that are common to species (modulating the expression and/or activity of a CDA). The specification provides no structural description of modulating the expression and/or activity of a CDA other than ones specifically exemplified; in essence, the specification simply directs those skilled in the art to go figure out for themselves what the claimed modulators looks like. The specification's disclosure is inadequate to describe the claimed genus of modulating the expression and/or activity of a CDA.

Applicant has disclosed only valsartan to associate with attenuation of CDA1 expression; therefore, the skilled artisan cannot envision all the contemplated modulator possibilities recited in the instant claims. Consequently, conception cannot be achieved until a representative description of the structural and functional properties of the claimed invention has occurred, regardless of the complexity or simplicity of the method. Adequate written description requires more than a mere statement that it is part of the invention. See *Fiers v. Revel*, 25 USPQ2d 1601, 1606 (CAFC1993). The Guidelines for the Examination of Patent Application Under the 35 U.S.C.112, ¶1"Written Description" Requirement make clear that the written description requirement for a claimed genus may be satisfied through sufficient description of a representative number of species disclosure of relevant, identifying characteristics, i.e., structure or other physical and or chemical properties, by functional characteristics coupled with a known or disclosed correlation between function and structure, or by a combination of such identifying

characteristics, sufficient to show the applicant was in possession of the genus (Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 20001, see especially page 1106 3rd column).

Vas-Cath Inc. v. Mahurkar, 19 USPQ2d 1111, makes clear that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention. The invention is, for purposes of the written description inquiry, whatever is now claimed." (See page 1117.) The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See Vas-Cath at page 1116.). Consequently, Applicant was not in possession of the instant claimed invention. See University of California v. Eli Lilly and Co. 43 USPQ2d 1398.

Applicant is directed to the final Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, ¶ 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

Legal Standard for Anticipation/Inherency Under - 35 USC § 102

To anticipate a claim under 35 U.S.C. § 102, a single prior art reference must place the invention in the public's possession by disclosing each and every element of the claimed invention in a manner sufficient to enable one skilled in the art to practice the invention. *Scripps Clinic & Research Foundation v. Genetech, Inc.,* 927 F.2d 1565, 1576, 18 U.S.P.Q.2d 1001, 1001 (Fed. Cir. 1991); *In re Donahue,* 766 F2d531, 533, 226 U.S.P.Q. 619, 621 (Fed. Cir. 1985). To anticipate, the prior art must either expressly or inherently disclose every limitation of the claimed invention. *MEHL/Biophile Int'l Corp. v. Milgraum,* 192 F.3d 1362, 1365, 52 U.S.P.Q.2d 1303, 1303 (Fed. Cir. 1999) (citing to *In re Schreiber,* 128 F.3d 1473, 1477, 44 U.S.P.Q. 1429, 1431 (Fed. Cir. 1997)); *Atlas Powder Co. v. Ireco Inc.,* 190 F.3d 1342, 1347, 51 U.S.P.Q.2d 1943, 1946 (Fed. Cir. 1999). To inherently anticipate, the prior art must necessarily function in accordance with, or include, the claimed limitations. *MEHL/Biophile*, 192 F.3d at 1365, 52 U.S.P.Q.2d at 1303. However, it is not required that those of ordinary skill in the art recognize the inherent characteristics or the function of the prior art. *Id.* Specifically, discovery of the mechanism underlying a known process does not make it patentable. See also MPEP §§ 2112, 2112.02 and 2145(II).

12. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e2) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

The changes made to 35 U.S.C. 102(e) by the American Inventors Protection Act of 1999 (AIPA) and the Intellectual Property and High Technology Technical Amendments Act of 2002 do not apply when the reference is a U.S. patent resulting directly or indirectly from an international application filed before November 29, 2000. Therefore, the prior art date of the reference is determined under 35 U.S.C. 102(e) prior to the amendment by the AIPA (pre-AIPA 35 U.S.C. 102(e)).

13. Claims 10-16 and 21 are rejected under 35 U.S.C. 102(b) as being anticipated by US. Pat. 6,211,217 as is evidenced by the specification on page 7, lines 18-20.

The `217 patent teaches that fibrous adhesions begin to form immediately following the surgical procedure and consist of collagen and other extracellular proteins (see col., 1, lines 56-58). The `217 patent teaches and claims a method for reducing fibrosis and adhesion formations in a surgical patient comprising administering a therapeutically effective amount of valsartan (AT_1 receptor antagonist which blocks the action of Angtensin II) prior to fibrosis and adhesion formation (see patented claim 1 and abstract), wherein the surgery is performed on an organ such as liver and kidney (see patented claim 3). Furhter the `217 patent teaches and claims a method for reducing fibrosis and adhesion formations in a patient with an induced pathological condition selected from the group consisting of post irradiation fibrosis comprising administering a therapeutically effective amount of valsartan prior to fibrosis and adhesion formation (see patented claim 9). The `217 patent further teaches that fibrosis can occur in any organ and accompanies many disease states such as hypertension (heart failure) and diabetes (nephropathy) (see col., 1, lines 15-20 in particular). While the `217 patent teachings is silent as to the "modulating the expression and/or activity of a CDA" per se; the method and the product used in the reference method are the same as the claimed method. Therefore "inhibiting modulating the expression and/or activity of a CDA" is considered inherent properties. As is evidenced in the specification on page 7 lines 17-20 discloses that therapeutic approaches which block the action of antiotensin II such as an AT1 receptor antagonist, valsartan, are associated with les cell injury and with attenuation of CDA1 expression. Accordingly, valsartan would modulate the expression and/or activity of a CDA.

The reference teachings anticipate the claimed invention.

14. Claim 10-16 and 21 are rejected under 35 U.S.C. 102(e) as being anticipated by US Pat. No.7,214,375 (of record), as evidence of Ozbun et al (Genomics 73:179-193 (2001) (see entire document) and the specification on page 8, lines 30-31 and page 12, lines 32-33.

The `375 patent teaches a method for treating or arresting the progress of pathologies characterized by an accumulation of extracellular matrix components by providing an agent to suppress the activity of transforming growth factor β (TGF-β) a peptide growth factor which is anabolic and leads to fibrosis and angiogenesis, wherein the agent is anti-TGF-β antibody (see col., 1, lines 54-60). The `375 teaches and claims a method of decreasing the deleterious accumulation of extracellular matrix (ECM) associated with a pathology or a condition wherein TGF-β-induced production and deleterious accumulation of extracellular matrix in a tissue exists comprising: providing an anti-TGF-β antibody that binds to TGF-β; and contacting the tissue

with the anti-TGF-β antibody that binds to TGF-β ; whereby the binding of the anti-TGF-β antibody to the TGF-β suppresses the deleterious accumulation of the TGF- β-induced extracellular matrix in the tissue, and wherein the pathology or condition is glomerulonephritis. (see claim 1). The `375 patent teaches that in the normal glomerulus, the mesangial cells are surrounded by extracellular matrix. An increase in the quantity of mesangial matrix, with or without mesangial hypercellularity, is the earliest histologic finding in many forms of glomerulonephritis and in diabetic nephropathy (see col., 4, lines 45-50).

The `375 teaches that TGF-β1 is known to increase extracellular matrix production (see col., 5, lines 49-51). As the instant specification defines TGF-beta as to increase CDA1 expression (see page 8, lines 30-31 and page 12, lines 32-33) thereby increase the production of ECM proteins such as fibronectin and collagen IV.

While the `375 patent is silence with respect to "modulating expression or activity" of a cell division auto antigen (CDA), Ozbun et al teach that DENTT (CDA) mRNA induction by TGF-β1 correlates with induction of TGF-β 1 mRNA, induction of extracellular matrix gene expression, and inhibition of colony formation in soft agarose in TGF-β1 responsive NSCLC cells when expressed to TGF- β1. Moreover, TGF- β1 does not induce DENTT mRNA expression in TGF-β1 nonresponsive NSCLC cells. Ozbun reported a novel TGF- β1 target gene with distinct domains for direction to different subnuclear locations (see abstract in particular). Accordingly, TGF-13 modulate expression and activity of CDA/DENTT.

The reference teachings anticipate the claim invention.

15. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

U.S. Patent No. 5,783,187 discloses a method for ameliorating a cell proliferative disorder by administering anti-CTGF antibodies. While the claims do not specifically recite the disorders of pending claim 11, patented claim 3 states that the disorder is "due to overgrowth of connective tissue cells", which is a functional description of fibrosis. As the instant specification defines CTGF as to increase CDA1 expression (see page 8, lines 30-31 and page 12, lines 32-33) thereby increase the production of ECM proteins such as fibronectin and collagen IV.

Eydelloth, et al., (WO 97/02032) disclose the use of converting enzyme (ACE) inhibitor and an angiotensin II receptor, (AII) antagonist in the treatment of a fibrotic condition at page 16, line 1, "interstitial fibrosis", which is associated with the tubulo-interstial changes of focal and segmental glomerulosclerosis (kidney fibrosis) (see last line of the abstract). The patient treated has elevated blood pressure (hypertension) because on page 3 of the `032 publication discloses that the co-administration of an ACE inhibitor with aII antagonist has been shown to be useful in the treatment of hypertension.

16. No claim is allowed.

17. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Maher Haddad whose telephone number is (571) 272-0845. The examiner can normally be reached Monday through Friday from 7:30 am to 4:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Eileen B. O'Hara can be reached on (571) 272-0878. The fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

October 10, 2008

/Maher M. Haddad/
Maher Haddad, Ph.D.
Primary Examiner
Technology Center 1600